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Executive–cognitive functioning in the development of antisocial personality disorder

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Abstract

The present study examined the association of cognitive–executive abilities to two risk factors for alcoholism, i.e., antisocial behaviors and a family history (FH+) of alcohol dependence. A sample of 91 right-handed, non-substance-dependent, young male adults recruited from the community were classified into three groups: (1) a control group of $n=32$ men with no history of DSM-III-R childhood conduct disorder (CD) or antisocial personality disorder (ASPD); (2) $n=25$ men who met criteria for a DSM-III-R childhood CD diagnosis, but did not meet diagnostic criteria for ASPD (i.e., CD/ASPD−); and (3) $n=34$ men who met DSM-III-R criteria for ASPD. They were further divided into those with and without a positive family history of alcoholism. A two-way (Antisocial Profile (3)×Family History of Alcoholism (2)) ANOVA was used to compare several neuropsychological measures of executive–cognitive functioning (ECF) ability. Verbal abstraction ability was found to be significantly lower in ASPD subjects compared with controls and CD-only subjects, inversely related to antisocial behavior severity (as measured by symptom count). CD-only and control subjects' abstraction ability were statistically indistinguishable. FH+ was associated with increased errors in planning performance on the Porteus Maze Test and diminished performance on Luria's simple alternate-tapping motor tasks. The effect was more pronounced when inhibition of prepotent motor planning was required. Results are consistent with previous work examining ECF ability in antisocial samples that find subtle differences in ECF ability compared to controls. The findings suggest that normal versus abnormal behavioral outcome for children with conduct problems may be influenced by cognitive ability profile, perhaps because of varying maturational processes.

Keywords: Antisocial personality disorder; Conduct disorder; Executive functioning; Cognitive ability; Family history

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1. Introduction

The consequences of childhood conduct disorder (CD) and its continuation into adulthood as antisocial personality disorder (ASPD) can represent tremendous societal and medical costs. CD and ASPD have been linked to various behavioral, social, and health problems, including aggression, criminal behavior, and substance disorder (Jordan, Schlenger, Fairbank, & Caddell, 1996; Lewis, Cloninger & Prais, 1983; Moeller, Doughtery, Lane, Steinberg, & Cherek, 1998; Patrick & Zempolich, 1998). Previous work highlights the role of CD and ASPD as a risk factor for substance dependence (Hesselbrock, Meyer & Hesselbrock, 1992), showing that adults with ASPD have a three- to fourfold greater likelihood of developing substance dependence compared to adults without ASPD. The widely noted association between alcoholism and ASPD has led to several theories proposing an ''antisocial alcoholic'' subtype (Babor et al., 1992; Cloninger et al., 1981; Jellinek, 1960), possibly with different etiological and developmental mechanisms than other subtypes.

Antisocial behavior shows heterogeneity of onset and expression across childhood and adolescence (Feehan, McGee, & Williams, 1994; Hinshaw, Lahey, & Hart, 1993; Lahey & Loeber, 1994; Lahey et al., 1995; Lahey et al., 1998; Loeber, 1991; Moffitt, 1993). These differences have complicated investigators' search to identify those factors that operate during maturation, which may be associated with ASPD. Longitudinal studies estimate at least one in four CD youth continues to display antisocial behaviors in adulthood (Robins, 1966). This raises the question of what factors operate to result in adult ASPD, or act to prevent the majority of problem-behavior youth from developing ASPD. A related question is whether the same factors also predispose youth to substance abuse and later alcohol dependence.

While environmental factors unquestionably play a role in the development of ASPD (Farrington, 1986), some researchers have examined neuropsychological test performance in an attempt to determine whether various neurobehavioral deficits predispose youth to ASPD. One approach has been to examine the relationship between executive–cognitive functioning (ECF) abilities and ASPD. ECF is thought to represent higher-order abilities involved in planning, initiation, and regulation of goal-directed behavior (Luria, 1980). ECF deficits in alcoholics include abstract reasoning, set shifting, set persistence, attention, verbal/categorical fluency, concept generation, persistence, temporal organization, sequencing, supervisory motor control, hypothesis formation testing, and cognitive flexibility (Giancola & Moss, 1998). The unifying element of these multifaceted abilities is that they are believed to have the majority of their primary neurological substrates in the prefrontal cortex (PFC) and its connections to other cortical and subcortical areas (Fuster, 1997; Luria, 1980).

Research has begun to address whether ECF ability in CD youth (Moffitt, 1993; Pennington & Ozonoff, 1996) or ASPD adults (Kandel & Freed, 1989; Gorenstein, 1987; Gorton, Swirsky-Sacchetti, Sobel, Samuel, & Goron, 1999) differs from healthy controls. In general, results favorably support the idea that deficits on neuropsychological tests of ECF can be found in these groups, as shown in a recent meta-analysis (Morgan & Lilienfeld, 2000). However, it remains unresolved if these deficits are solely attributable to executive function, or if impairments of other neuropsychological abilities are implicated. Despite the encouraging results, no published studies to date have examined whether ECF ability varies

with changes in antisocial behavior between adolescence and early adulthood. Such work would address the hypothesis that ECF impairment is associated with the onset and/or stability of disruptive problem behavior. Previously, we investigated this issue by examining relationships between ECF ability and CD/ASPD diagnoses for substance-dependent inpatients (Stevens et al., in press). We found that patients who met the criteria for ASPD had lower vocabulary and verbal abstraction ability compared to both normal controls and to patients who had CD as children, but not ASPD as adults. However, we did not control for possible attention-deficit hyperactivity problems. It has been suggested that ECF deficits in CD groups may only be present in comorbid CD/ADHD groups (Pennington & Ozonoff, 1996).

When considering the relationship of ECF ability and CD/ASPD, an important related issue is the risk for substance dependence inherent in antisocial behavior disorders. Giancola and Moss (1998) suggest that ECF deficits are the most consistent and predominant of all the cognitive deficits found in alcoholics. These abilities are believed to be related to processes of reinforcement, learning, and motivation (Fuster, 1989; Iversen & Mishkin, 1972; Rolls, Hornak, Wade & McGrath, 1994), which may influence the development of substance use. Predicated on presumed biological predisposition to alcoholism (Schuckit, 1987; Tarter, 1988), studies have examined ECF ability in both youth at-risk for alcohol disorder because of a positive family history of alcoholism (Schuckit, 1987). Reviews of early studies showed that the evidence for a relationship between ECF and a positive family history (FH+) of alcoholism was mixed (Schuckit, 1987; Searles, 1990; Tarter, Laird, & Moss, 1990). ECF deficits found in ASPD adults (Gorenstein, 1987) and CD youth (Moffitt, 1993), along with electrophysiological evidence (Bauer, 1997; Bauer & Hesselbrock, 1993; Bauer, O'Connor, & Hesselbrock, 1994; O'Connor, Bauer, Tasman, & Hesselbrock, 1994) are consistent with some investigators' suggestion that the cognitive performance differences found in earlier FH+ studies are likely due to a high prevalence of CD and/or ASPD in the samples, rather than family history status (Gillen & Hesselbrock, 1992; Malloy, Noel, Rogers, Longabaugh, & Beattie, 1989). The etiological relationships among, FH+, CD/ASPD, and ECF have yet to be disentangled.

The field of developmental neuropsychology has emerged in recent years to examine cognitive ability from a perspective that includes central nervous system maturation. Research has emerged to characterize cognitive development in various groups of youth, using studies of human (Diamond & Goldman-Rakic, 1985), animal (Diamond, 1988; Diamond, Zola-Morgan, & Squire, 1987), and psychopathological youth whose abnormal behavior is thought to stem from suspected or identified brain injury, or problems with brain maturation (Cattelani, Lombardi, Brianti, & Mazzucchi, 1998; Pennington & Ozonoff, 1996). This research not only seeks to describe the development of specific cognitive abilities, but also the development of different brain systems and their changes at later ages (Welsh & Pennington, 1988).

Our current understanding of ECF development posits that age-appropriate forms of ECF ability are present in early childhood, but then ECF shows a protracted course of full maturation (Welsh & Pennington, 1988), extending into late adolescence and early adulthood. In this context, it is plausible that a developmental ECF abnormality may

underlie antisocial behavior in some groups of youth and adolescents. Although others have speculated that deficits or maturational lags in self-governing ECF ability development may influence the expression of problem behavior in some youth (Hill, 1999; Hogan, 1999; Smith, Kates, & Vriezen, 1992), it is not known whether ECF abilities causally influence the expression of antisocial behavior. This is partly because of our incomplete understanding of frontal lobe maturation and ECF development (Smith et al., 1992).

The present study examined whether remission of antisocial behavior after adolescence is associated with normal ECF development, when compared with normal controls and youth whose antisocial behaviors do not remit. The study has three objectives: First, the study used formal diagnostic classification criteria (i.e., CD and ASPD diagnoses determined from retrospective self-report) to examine whether adult executive–cognitive abilities differ according among three profiles of antisocial behavior development (i.e., young adults with no history of antisocial behavior, CD-only subjects, or subjects with ASPD). It was predicted that young adult ASPD+ subjects would show lower ECF ability than either normal controls or CD-only subjects, while CD-only and controls would be comparable. Second, we were interested in whether FH+ enhances or reduces neuropsychological test performance among the three antisocial behavior groups. Third, to account for the possible confound of childhood ADHD, correlational analyses examined the relationships between ECF ability and disruptive behavior severity occurring prior to age 12.

2. Method

The study represents secondary analyses of data collected for a larger project investigating several risk factors for alcoholism, including family history of alcoholism and personal history of ASPD. Data from this study have been previously presented (Bauer et al., 1994, Hesselbrock & Gillen, 1992), and the experimental protocol is described in greater detail in these papers.

2.1. Participants

Participants in this study included 91 right-handed, Caucasian males aged 21–28. They were recruited from the Hartford/New Haven area by way of newspaper, radio, and television advertisements following the methodology proposed by Widom (1978). Advertisements sought persons who characterized themselves as thrill-seeking, adventurous, or carefree. Respondents were screened via telephone interview for obvious substance abuse, maternal history of drinking during pregnancy, history of psychosis, medical/neurological problems, and significant head injury (i.e., loss of consciousness greater than 24 h). Potential participants were also excluded if they were female, left- or mixed-handed, or were taking any prescription or over-the-counter medications. Informed consent was obtained from all subjects prior to participation. Participants were paid US$50 upon completion of the study.

2.2. Procedure

The NIMH Diagnostic Interview Schedule (DIS) version III-A (Robins et al., 1981) was administered by trained research assistants. The DIS yielded DSM III-R (American Psychiatric Association, 1987) diagnoses of current and lifetime substance-related and psychiatric illnesses, including childhood CD and ASPD. No participant was currently depressed or dependent on alcohol or other drugs, as determined by the DIS. The criteria for ASPD were modified to exclude those symptoms that were present in the context of alcohol or substance use (e.g., disorderly conduct, driving while intoxicated, etc.). Based on diagnoses of CD and ASPD provided by the DIS, the participants were further classified into three antisocial behavior profiles: (1) those with no history of CD or ASPD (i.e., normal controls, $n=32$); (2) those with a history of CD, but whose present symptoms do not meet diagnostic criteria for ASPD (i.e., CD/ASPD−, $n=25$); and (3) those who met criteria CD and have current diagnosis of ASPD (CD/ASPD+, $n=34$).

Family history of alcohol use problems were obtained from each subject using an interview constructed by Andreasen et al. (1977). A participant was considered to have a positive family history of alcoholism according to FH-RDC criteria (Spitzer, Endicott & Robins, 1975) if either his biological father or a sibling plus an additional biological second degree male relative (e.g., grandfather or uncle) had a history of alcoholism. Subjects with a maternal history of alcoholism were excluded. No control subject reported a history of alcoholism in any first or second degree biological relative.

Data were also collected on alcohol consumption, age of onset for alcohol and drug use, and the Michigan Alcoholism Screening Test (MAST) (Selzer, 1971). Behavioral ratings of attention-deficit hyperactivity symptoms were obtained using items selected from previous behavioral checklists (Tarter, McBride, Buonpane, Dorothea, & Schneider, 1977; Wender, 1971) querying behavior prior to age 12. More information on these scales is available in Hesselbrock (1986).

2.3. Neuropsychological test measures

A battery of neuropsychological instruments was administered to each subject by trained research technicians. The tests administered in the study were chosen to assess a broad range of cognitive abilities, including general intellect (IQ), memory, motor skills, visuospatial ability, and measures assessing ECF. The test instruments selected for the current analysis have demonstrated relationships to anterior brain functioning and/or ECF (Pennington & Ozonoff, 1996).

2.3.1. Luria Motor Tasks (Golden, Purisch, & Hammeke, 1985)

Measures included two alternate tapping tests (LMT No. 22 and No. 23) in which the number of times participants produced a pattern of taps (right finger twice then left once, followed by another trial reversing the order) within 10 s.

2.3.2. Trail-Making Test (Reitan, 1979)

Participants must connect consecutive numbered and lettered circles by alternating between the two sequences as quickly as possible without errors. This test is believed to

access mental set-shifting ability. The dependent measure is the total time in seconds to complete the task.

2.3.3. Controlled Oral Word Association Test (Benton & Hamsher, 1978)

The COWAT consists of three word-generation trials during which participants are asked to quickly name words that begin with the letters C, F, and then L. The dependent measure is the sum of the number of words generated on all three trials.

2.3.4. Wechsler Adult Intelligence Scale-Revised (Weschler, 1981)

The dependent measures used in the current study include Similarities, Information, Digit Span, and Picture Arrangement subtest raw scores. The Information subtest is used as part of a control for academic achievement, while the other three scores represent aspects of information processing thought to be involved in executive function (i.e., verbal abstraction, working memory, and mental sequencing).

2.3.5. Wisconsin Card Sorting Test (Berg, 1948; Grant & Berg, 1948)

The WCST requires participants to match cards with combinations of different numbers of variously colored shapes to categorical exemplars. The current analyses examined two dependent measures, Total Categories achieved within 128 trials (i.e., concept formation), and Perseverative Errors (i.e., cognitive inflexibility). Administration rules for the WCST are detailed in Heaton (1981).

2.3.6. Porteus Maze Test (Porteus, 1965)

Participants use a pencil to trace paths through a series of progressively more difficult untimed mazes without entering any blind alleys and without lifting their pencil. The dependent measure used is the highest mental age year achieved without error.

2.4. Analyses

Neuropsychological measures of ECF were compared using two-factor univariate analysis of variance for the (n=91) sample. Antisocial profile (3) and family history of alcohol dependence (2) were the design factors. Before conducting these analyses, the possible effect of Verbal IQ on neuropsychological test scores was examined as a covariate in a series of ANCOVAs using antisocial profile as the single design factor and each neuropsychological test score as the dependent variable. Verbal IQ significantly covaried with only two dependent variables, Trails B and Porteus Maze highest year achieved without error. For these two dependent variables, Verbal IQ was used as a covariate. All other cognitive test scores were unaffected by differences in Verbal IQ.

Because it was predicted that the normal control and CD-only groups would be similar in relation to ECF ability but differ from the ASPD group, these analyses used planned comparisons to examine differences among the groups. We also decided to employ planned comparisons to maximize sensitivity. Theoretical differences among abilities measured by the various ECF tasks made it unlikely that we would find multivariate differences among groups.

The second set of analyses employed use of zero-order Pearson correlation coefficients to examine the linear association of both child and adult problem behavior and attention-deficit disorder symptom severity with neuropsychological test performance.

3. Results

3.1. Demographic information for antisocial profiles

The demographic features, general intelligence estimates, and monthly alcohol consumption of the three antisocial groups were compared. No significant differences among the three groups for age, education, WAIS-R Performance IQ score, MAST score, or average amount of absolute alcohol consumed in the past 6 months. WAIS-R Verbal IQ was significantly different ($P=.036$) among the three groups. Post hoc testing revealed that only normal control subjects differed from CD/ASPD−. The means, standard deviations, percentages, and significant differences among groups are displayed in Table 1.

3.2. Antisocial profile and ECF results

The means, standard deviations, and significance levels for comparisons of ECF test performance for each diagnostic classification category are displayed in Table 2. There was a significant main effect of antisocial profile for WAIS-R Similarities [$F(2,79)=4.171$, $P<.05$].

Trend levels of significance were found for WAIS-R Digit Span [$F(2,79)=2.755$, $P<.10$], Picture Arrangement [$F(2,79)=2.442$, $P<.10$], and for Luria Motor Task No. 22 Extraneous Errors [$F(2,79)=2.579$, $P<.10$]. No other ECF measure was significantly different across antisocial groups.

Planned comparison results indicate that the difference in Similarities scores is primarily due to the significant difference ($P<.01$) between normal controls and CD/ASPD+. A

Table 1
Demographic differences by antisocial behavior classification

Variable	Normal controls ($n=32$)	CD/ASPD− ($n=25$)	CD/ASPD+ ($n=34$)	P
	Mean (S.D.)	Mean (S.D.)	Mean (S.D.)	
Age (years)	22.5 (1.34)	23.2 (1.53)	23.4 (1.75)	n.s.
Education (years)	15.4 (1.31)	15.3 (1.57)	14.7 (1.55)	n.s.
WAIS Verbal IQ	113.0 (12.97)	104.1 (13.36)	107.5 (12.53)	.036
WAIS Performance IQ	105.4 (14.54)	100.0 (13.69)	103.0 (11.19)	n.s.
MAST score	3.2 (3.25)	4.2 (2.97)	5.9 (6.55)	n.s.
Alcohol last 6 months (oz.)	45.5 (44.9)	60.0 (80.0)	82.3 (103.2)	n.s.
Paternal alcoholism history	46.4%	50.0%	41.4%	n.s.

All continuous variables used one-way ANOVA to test for significant differences. Paternal alcoholism history differences tested by Pearson chi-square.

significant linear effect was detected across the groups with Similarities score decreasing as antisocial severity increased. Planned comparisons showed that CD/ASPD− subjects had significantly lower Digit Span performance than normal controls ($P<.05$) or ASPD+ subjects ($P<.05$). There was no statistical difference between normal controls and ASPD+ subjects on Digit Span scores. A similar pattern was found for Picture Arrangement scores. There was a significant difference between normal controls and CD/ASPD− ($P<.05$) and a trend difference between CD and ASPD+ ($P<.10$) for Picture Arrangement scores. Furthermore, there was no difference between ASPD+ and normal controls on Picture Arrangement scores.

The mean number of LMT No. 22 Extraneous errors showed a significant difference ($P<.05$) between CD/ASPD− and CD/ASPD+ subjects, but not for any other pairs of contrasts.

3.3. Family history and ECF results

Table 2 also indicates significant effects for FH on ECF test results. In the analysis of covariance controlling for the influence of Verbal IQ, a main effect of family history status was found for Porteus Maze highest year without error score, $F(1, 82)=4.128$, $P<.05$. This Porteus Maze score was higher for family history negative subjects by an average of approximately 2 years in each antisocial profile category. Trails B also tended to be different [$F(1,82)=2.869$, $P<.10$]. No other ECF measure was significantly different between FH groups.

Table 2
ECF by antisocial behavior classification

Variable	Controls ($n=32$)	CD/ASPD− ($n=25$)	CD/ASPD+ ($n=34$)	P
	Mean (S.D.)	Mean (S.D.)	Mean (S.D.)	
WAIS Similarities*	22.4 (2.78)	21.3 (2.70)	19.9 (4.08)	.019
WAIS Information	22.2 (4.32)	20.2 (40.03)	21.7 (3.53)	n.s.
WAIS Digit span*	19.3 (4.21)	16.9 (4.02)	19.2 (4.16)	.070
WAIS Picture Arrangement*	14.7 (2.08)	12.8 (2.97)	14.3 (2.91)	.094
WCST Categories achieved[a]	5.4 (1.41)	5.5 (1.22)	5.6 (1.03)	.028
WCST Perseverative Errors	11.2 (8.75)	8.5 (5.46)	10.7 (7.18)	n.s.
Porteus highest year without error[b]	10.8 (3.25)	9.2 (4.86)	9.5 (4.08)	.045
Trails A[a]	20.8 (4.40)	22.5 (9.51)	21.2 (5.83)	.031
Trails B[b]	46.4 (24.21)	48.6 (15.87)	50.0 (15.00)	.094
Verbal fluency	55.6 (7.42)	54.5 (9.43)	54.6 (9.42)	n.s.
LMT No. 22 Tap right hand twice	19.1 (5.94)	17.8 (5.65)	17.2 (5.82)	n.s.
LMT No. 22 Extraneous errors*	0.9 (1.34)	0.7 (0.92)	1.5 (1.81)	.082
LMT No. 23 Tap left hand twice[a]	16.8 (5.34)	14.8 (5.13)	15.6 (4.99)	.021
LMT No. 23 Extraneous errors	0.8 (1.13)	1.1 (1.26)	1.2 (1.65)	n.s.

[a] CD/ASPD×Family History interaction effect at least $P<.10$ (means not shown).
[b] Family history effect at least $P<.10$ (means not shown).
* CD/ASPD effect at least $P<.10$.

3.4. Antisocial Profile×Family History interaction

The interaction of Antisocial Profile and FH was examined in relation to ECF. Significant interactions were found for three ECF measures. Trails A completion time revealed a significant Antisocial Profile (3)×FH (2) interaction [$F(2,79)=3.619$, $P<.05$]. Total Categories achieved on the WCST also showed a significant interaction [$F(2,79)=4.154$, $P<.05$]. There was a significant interaction for Luria Motor Task No. 23 (tap left hand twice/right hand once) [$F(2,79)=4.084$, $P<.05$]. These significant interactions are also indicated in Table 2.

Therefore, each of the components of these 3×2 interactions were retested as a series of three 2×2 interaction contrasts. For Trails A completion time, the significant components of the FH×Antisocial Profile interaction were between normal controls and CD/ASPD+ [$F(1,60)=3.278$, $P<.10$] and between CD/ASPD− and CD/ASPD+ [$F(1,53)=6.196$, $P<.05$] (Fig. 1). For WCST Total Categories, there was a significant interaction between normal controls and CD/ASPD− [$F(1,53)=3.845$, $P<.10$] and between CD/ASPD− and CD/ASPD+ [$F(1,52)=9.000$, $P<.01$] (Fig. 2). For LMT No. 23, interaction component was significant between normal controls and CD/ASPD+ [$F(1,60)=7.619$, $P<.01$] and between CD/ASPD− and CD/ASPD+ [$F(1,53)=4.290$, $P<.05$] (Fig. 3).

3.5. Association of disruptive childhood behavior with ECF

In order to determine whether various disruptive behaviors influenced test scores, the intercorrelation of neuropsychological test scores and symptoms counts of childhood hyperactivity, impulsivity, inattention, and childhood and adulthood conduct problems were computed (see Table 3).

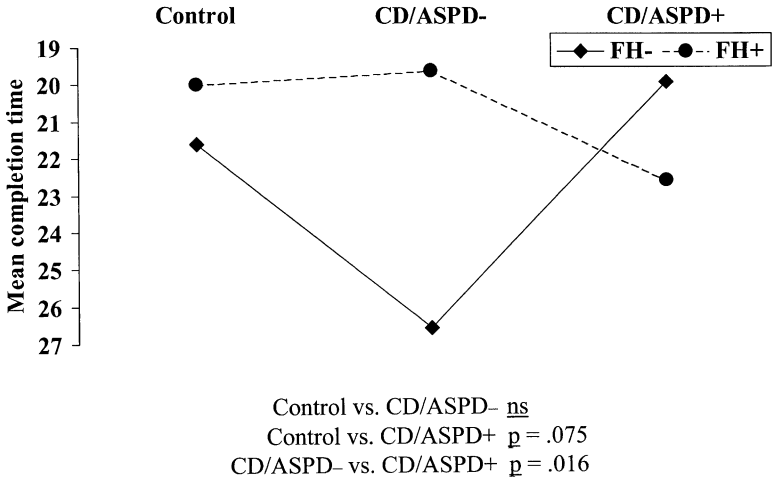
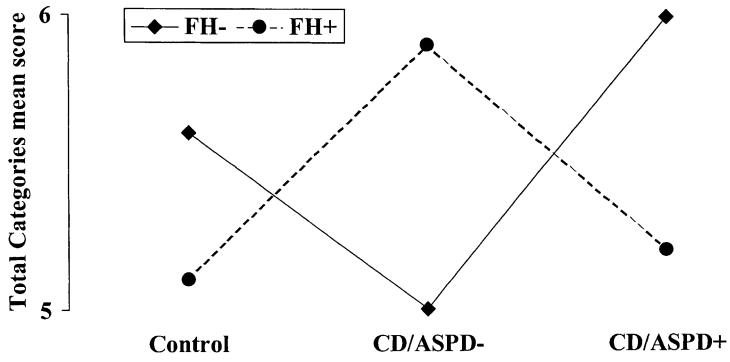


Fig. 1. FH×Antisocial Behavior Classification interaction: Trails A (s).

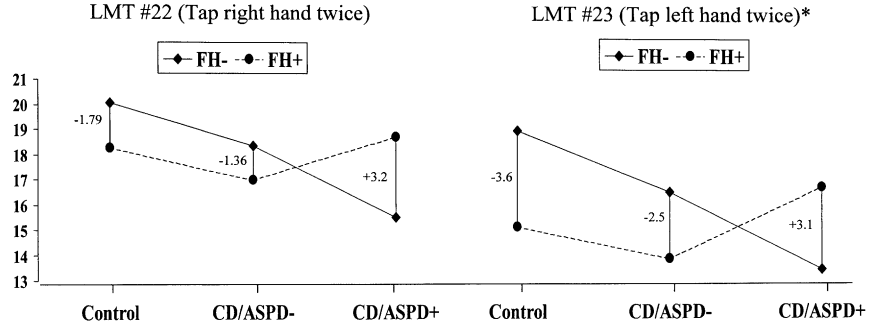


Control vs. CD/ASPD– p = .055
Control vs. CD/ASPD+ ns
CD/ASPD– vs. CD/ASPD+ p = .004

Fig. 2. FH×Antisocial Behavior Classification interaction: WCST Total Categories achieved.

Significant associations were found between Impulsivity and WAIS-R Full Scale IQ, Verbal IQ, and the WAIS Information and Picture Arrangement subtests. A significant association was also found between Inattention symptoms and WAIS-R Verbal IQ, Information, and Picture Arrangement.

Hyperactivity was significantly associated with Verbal IQ and Luria Motor Task No. 22 (tap right hand twice/left hand once). Childhood CD symptom count severity was uncorrelated to adult ECF performance. However, the number of adult ASPD symptoms was associated with WAIS-R Similarities scores. As would be expected, Impulsivity,



(Overall Interaction ns)

(Overall Interaction p < .05)
Control vs. CD/ASPD– ns
Control vs. CD/ASPD+ p = .008
CD/ASPD– vs. CD/ASPD+ p = .043

Fig. 3. FH×Antisocial Behavior Classification interaction: Luria Motor Task comparison.

Table 3
Zero-order correlation of ECF test measures with disruptive behavior symptoms

	Impulsivity	Inattention	Hyperactivity	CD symptoms	ASPD symptoms
WAIS Full Scale IQ	−.264*	−.204	−.196	−.180	−.026
WAIS Verbal IQ	−.303**	−.292**	−.276**	−.207	.033
WAIS Similarities	−.164	−.206	−.203	−.199	−.275**
WAIS Information	−.249*	−.306**	−.225*	−.182	.075
WAIS Digit Span	−.014	−.202	−.048	−.041	.197
WAIS Picture Arrangement	−.330**	−.223*	−.181	−.112	.060
WCST Categories achieved	−.039	−.138	−.049	.037	−.009
WCST Perseverative Errors	.009	.120	.140	−.060	−.035
Porteus highest year without error	.200	.151	.081	−.096	−.043
Trails A	−.005	.058	.099	.089	.195
Trails B	.127	.113	.082	.105	−.020
Verbal fluency	−.038	−.001	.012	−.048	.028
LMT No. 22 Tap right hand twice	−.095	−.062	−.212*	−.101	.039
LMT No. 23 Tap left hand twice	−.031	−.110	−.092	−.175	.081
Impulsivity	1.000	.515**	.658**	.394**	.139
Inattention	.515**	1.000	.567**	.300**	.116
Hyperactivity	.658**	.567**	1.000	.405**	.189
CD symptoms	.3936**	.300**	.405**	1.000	.393**
ASPD symptoms	.139	.116	.189	.393**	1.000

* $P<.05$.
** $P<.01$.

Inattention, and Hyperactivity were all significantly intercorrelated. Childhood CD symptoms were also significantly intercorrelated with these problem behavior symptoms counts. However, adult ASPD symptom count was correlated only to the number of childhood CD symptoms. These findings suggest that childhood behaviors indicative of ADHD might have been a factor in neuropsychological performance for at least some of the subjects, but was not a widespread confound.

4. Summary and discussion

The present study examined whether remission of childhood conduct problem behaviors is associated with normal ECF ability in adulthood. All three groups displayed average or above average general cognitive ability. In accordance with our primary hypothesis, small differences in verbal abstraction ability significantly differentiated ASPD+ subjects from normal controls and CD-only subjects. Furthermore, verbal abstraction scores generally decreased with greater number of adult antisocial behavior symptoms. Within the current sample, this variable appears to depict a mostly resolved developmental delay in cognitive development. The overall high cognitive and functional level of these subjects suggest that verbal abstraction deficits in this group are subtle, and may merely represent the low end

of the normal range of functioning. Subtle deficits, however, often have profound effects on behavior and overall functionality. Other measures of ECF ability did not follow our predictions for antisocial profile, and no clinically meaningful differences emerged for their effects.

FH+ compared to FH− subjects made increased errors in successful planning performance on the Porteus Maze Test. However, performance on less cognitively complex tests, which require less of a cognitive load, was not affected by FH status (e.g., simple psychomotor tests like Trails A or the Porteus Maze age equivalency that does not take performance errors into account). Worsened performance as a result of higher cognitive load through task complexity is consistent with theoretical predictions of ECF dysfunction (Baddeley & Della Salla, 1998). The failure in previous work to find ECF differences related to FH+ in part may be related to the lack of such an increased load in tasks utilized in those studies. The interaction of FH and ASPD on one of the Luria motor tasks suggests that FH+ had a negative effect on sustained alternating motor performance for at least two of the three groups (normal controls and CD/ASPD− groups). This negative effect is doubled on the task where subjects are also required to inhibit prepotent motor programming (i.e., switch to reciprocal task). Furthermore, the interaction of ASPD+ and FH+ resulted in relatively better performance in FH+ subjects compared to FH− subjects. This result is not consistent with our theoretical expectations. It is tempting to speculate ways that FH+ and ASPD+ act in concert to influence the developing CNS system. However, the lack of coherent patterns in the data suggests that this finding most likely represents sampling error, which for neuropsychological data, most commonly occurs because of motivation problems or fatigue.

The ECF findings for different antisocial profiles represent an avenue of exploration that is consistent with previous research. Studies of psychopaths and violent criminals (Fedora & Fedora, 1983; Gorenstein, 1982; Yeudall & Fromm-Auch, 1979) have revealed differences in presumed ECF test performance — primarily in WCST and Trail-Making. The current study relies on DSM-III-R criteria rather than psychopathy checklists, and may account for the emergence of a verbal abstraction difference for the current ASPD sample. In addition, it should be reiterated that the current sample comprises functional adults with higher-than-average intelligence. Studies of IQ suggest that intelligence does play a role in onset and stability of antisocial behavior into adulthood (Farrington, 1991; Lahey et al., 1995), but only for low-IQ subjects. While one can question the association between low IQ and antisocial behavior stability, the literature lacks well-controlled longitudinal studies of ECF functioning in CD samples.

The presence of subtle ECF differences for these high-functioning males with average or above-average intelligence potentially increases the likelihood of greater risk for impaired functioning if they sustain even mild additional central nervous system insult to anterior brain regions (e.g., as the result of head trauma, alcoholism, etc). For example, it would be interesting to discover whether brain injury recovery in this average-intelligence ASPD group is worse when compared to non-ASPD subjects with comparable IQs. These differences deserve further study, using a broad range of empirically validated tests sensitive to prefrontal dysfunction in childhood and adolescence. Such work may yield improved understanding of the cognitive underpinnings of ASPD, as well as the interrelationship of various executive–

cognitive abilities. Such knowledge also will likely lead to better understanding of factors that lead to the higher prevalence of substance dependence in this group.

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